SPOTLIGHT ON PUBLIC HEALTH RESEARCH

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A Public Health Problem: Consequences of Trauma on Health Outcomes and the Role of Social Support

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Abstract

Introduction: Extensive research substantiates a negative correlation between stress and health. The implications of traumatic stress are complex, affecting the physical, psychological, physiological, and social health of individuals. The aim of this study was to examine the role of social support in relation to trauma-related health consequences.

Methods: Nationally representative data were obtained from the Midlife in the United States study, covering the period 2004-2006, and used in regression models to predict the relationships between types of trauma (adult vs. childhood), measures of social support, and biomarkers of stress reactivity (cortisol, high sensitivity C-reactive protein (CRP), and number of health conditions).

Results: The study found that an increase in traumatic experiences during adulthood was associated with a higher logged cortisol level, but social

support did not buffer these effects. No significant trends were observed with childhood trauma.

Conclusions: Results suggest the importance of addressing indicators from multiple domains simultaneously to investigate the effects of trauma and social support on biomarkers of stress.

Keywords: social support, trauma, cortisol, high sensitivity C-reactive protein, health conditions

Introduction

The economic cost of health care imposed on society and attributed to life stressors and trauma-related outcomes is substantial. The United States Centers for Disease Control state that trauma-related outcomes are a public health problem (Sleet, Dahlberg, Basavaraju, Mercy, McGuire, & Greenspan, 2011). When individuals in society experience cumulative and chronic individual life stressors, these factors influence later life health outcomes (Mielock, Morris, & Rao, 2017; Sapolsky, 2004) and mortality rates (Holt-Lunstad, Smith, & Layton, 2010) The odds of mortality increase by 91% for individuals who are socially isolated, with a 50% increase in the odds of death among individuals who lack social connections (Holt-Lunstad et al., 2010).

Individuals in society who are exposed to prolonged stress and trauma experience anxiety (O'Donovan, Slavich, Epel, & Neylan, 2013) and depression (Heim, Mletzko, Purselle, Musselman, & Nemeroff, 2008). Psychosocial implications of trauma, especially intimate partner violence,

and stress on health include homelessness and lack of access to necessary services (Breiding, Chen, & Black, 2014). For aging individuals, trauma and life stressors alter sensitivity of immune cell receptors, thus increasing risks for disease (Williamson, Porges, Lamb, & Porges, 2015). Previous studies have shown a relationship between stress, anxiety, and inflammation (Barr, 2014; Kubzansky, Seeman, & Glymour, 2014; Riancho & Brennan-Olsen, 2017), which will lead to poorer health outcomes.

Evidence-based research associates concerns about the population's health with trauma, life stressors, and cortisol; however, other factors such as the state of an individual's mental health (Carpenter, Shattuck, Tyrka, Geracioti, & Price, 2011) and genetics (Gillespie, Phifer, Bradley, & Ressler, 2009) contribute to influencing how individuals react to life stressors, and, in particular, trauma (Bosch, Riese, Reijneveld, Bakker, Verhulst, Ormel, & Oldehinkel, 2012). Cortisol serves as a measurement of an individual's response to stress and perceived threats. The use of cortisol as a biomarker of inflammation in research related to stress and trauma is well documented (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011; Bosch et al., 2012; Carpenter et al., 2011; Suzuki, Poon, Papadopoulos, Kumari, & Cleare, 2014) because the consequences of elevated cortisol levels due to stress and trauma are a major concern. Similarly, CRP is an inflammatory marker that may indicate the development of cardiovascular disease (CVD) (Alley, Seeman, Kim, Karlamangla, Hu, & Crimmins, 2006), among other health conditions. A body of research postulates that CRP is elevated in the body

when inflammation is present, and increases as the level of inflammation also increases. The current study assessed CRP, along with cortisol, as indicators of stress reactivity because they are easy biomarkers to obtain in clinical settings, in particular CRP as collected through point-of-care procedures.

Research correlates a history of early life stressors and trauma commencing in childhood to longstanding effects on health in adulthood (Bosch et al., 2012; Suzuki et al., 2014). The effects of stress mediated by trauma additionally predispose individuals to psychiatric disorders (Tyrka, Wyche, Kelly, Price, & Carpenter, 2009). In adults, responses to psychological stress tasks measured by salivary cortisol are higher in men compared to women (Lovallo, Farag, Vincent, Thomas, & Wilson, 2006). Comparably, men showed decreased salivary cortisol levels, depending on the quality of social support (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995). However, higher cortisol responses to stress tasks were found in women supported by their own life partners (Kirschbaum et al., 1995). The effects of social support on individuals following trauma are reported to mediate life's stressors. Social support in the form of friends, family, or a spouse, whether the support be formal or informal, displays powerful buffering effects on trauma (Chin, Murphy, Janicki-Deverts, & Cohen, 2017; Uchino, Cacioppo, & Kiecolt-Glaser, 1996) when compared to the inflammatory slopes of individuals with limited social support (Chin et al., 2017; Heinrichs et al., 2003), including

individuals who have been socially isolated (Yang, McClintock, Kozloski, & Li, 2013).

Evaluations of adult trauma, social support, and childhood maltreatment indicated participants' lack of perceived social support was found to be the single strongest predictor of the development of PTSD symptoms (Evans, Steel, & DiLillo, 2013). When individuals perceive social support to be available, positive effects are seen for mental health as well as for self-reported general health (Ke, Liu, & Li, 2010). Increased social support is also associated with lower cortisol levels and a decreased risk for stress-related psychopathology (Uchino et al., 1996). The studies discussed above serve as potential evidence of the importance of a social support system following trauma in mediating the consequences of lifelong stress on an individual's health. The response to early life stressors and trauma is also demonstrated through inflammation in the body and measured via increased cortisol and CRP, along with lifelong psychological, physiological, and psychosocial implications, which forms a significant basis for research that highlights the importance of a focus on the role of social support in mediating life's stressors (Ozbay, Johnson, Dimoulas, Morgan Iii, Charney, & Southwick, 2007).

This study takes an interdisciplinary approach to assess the associations between stress, inflammation (as measured by cortisol, CRP, and number of health conditions), social relationships, and health. Thus, we examined the consequences of trauma on the health of human participants

using two hypotheses. We hypothesized first that, net of potential social and biological confounders, trauma in childhood and adulthood would be positively associated with cortisol levels, in midlife whereas social support from a friend, family, or a spouse during midlife would be negatively associated with cortisol levels. Second, we hypothesized that, when taking into account potential social and biological confounders, trauma in childhood and adulthood would be positively associated with risk of high CRP and number of chronic health conditions at midlife, whereas social support at midlife would be negatively associated with risk of high CRP and number of chronic health conditions.

Methods

Participants and Procedure

Data came from Midlife in the United States (MIDUS) Wave 2. MIDUS began in 1994-1995 as a survey of 7,108 participants in America aged 25 to 74. The MacArthur Midlife Research Network conducted the study to provide a comprehensive dataset for researchers interested in examining psychosocial and biological data (MIDUS, 2011). MIDUS 2 was collected in 2004-2006 as a follow-up to MIDUS and included five projects and a Milwaukee sample. The current study used data from Project 1 and Project 4 of MIDUS 2 collected in the same time frame. We excluded the Milwaukee sample because those data are not available in the public-use data set. Project 1 included a survey assessing psychosocial, sociodemographic, and health variables, and Project 4 contained biomarker assessments on a

subsample of Project 1 participants. Fasting blood, 12-hour urine, and saliva samples were collected for biomarker data at three General Clinic Research Centers including University of California, Los Angeles, University of Wisconsin, and Georgetown University (Dienberg Love, Seeman, Weinstein, & Ryff, 2010). The results of these tests, processed at the time of data collection, are available in the public use data set.

MIDUS 2 data provided comprehensive indirect observation (self-report) indicators as well as direct observation (biomarker) indicators from a follow up of 1,255 original MIDUS respondents. The self-reported data included psychosocial and behavioral assessments, while the biomarker data (1,054 participants) included biological assessments, all of which allowed for examination of stress implications and health outcomes. The sample included respondents ages 34 to 84 that participated in both the survey and the biomarker project of MIDUS 2. The sample was restricted to respondents who gave samples for both salivary cortisol and CRP, and had no missing values for all dependent variable data. The total remaining sample size was 973. Appendix Table 1 provides a comparison of the analytic sample characteristics and the characteristics of the full biomarker sample.

Measures

Three dependent variables were used to test the hypotheses. The first dependent variable was the log of the average of the baseline salivary cortisol measures, measured in nanomoles per liter (nmol/L). Each baseline

measure was taken from the biomarker saliva assays, and then MIDUS created a variable that is the average of these measures. Participants provided saliva samples at four time points a day over a period of four days: 1) when they awoke, 2) 30 minutes after they awoke, 3) before lunch, and 4) before bedtime. Saliva samples were collected on cotton swabs and frozen until the time of the assays where the concentration of free cortisol was detected. The waking measures constitute the baseline measures. Measuring cortisol allowed for the assessment of neuroendocrine reactivity and contributed to further knowledge of the implications of traumatic stress pertaining to bodily systems.

The second dependent variable was CRP, measured in micrograms per deciliter (μg/dL). CRP data were taken from biomarker blood assays performed at the Laboratory for Clinical Biochemistry Research in Vermont. Fasting blood samples were collected on the second day of participants' hospital stays (which facilitated the early morning, fasting blood draws) and were all processed at the General Clinical Research Center (GCRC) with standardized procedures to ensure consistency. To determine high CRP, we constructed a binary measure, equal to 1 if CRP was >=3 and <10, and equal to 0 if less than 3. Individuals with CRP levels greater than 10 were dropped from analysis as this may indicate an underlying condition (Pearson, Mensah, Alexander, Anderson, Cannon, Criqui, Fadl, Fortmann, Hong, & Myers, 2003).

The third dependent variable was number of chronic health conditions. Chronic health conditions may be the long-term outcome of inflammatory processes in the body. Because this variable is unevenly distributed, we created a three-category measure: zero chronic conditions, one chronic condition, and two or more chronic conditions.

Although our primary interest was in how trauma and social support might be associated with inflammation, one of the limitations of prior literature is that it has not thoroughly examined these factors in the presence of a wide range of potential confounders, which can lead to biased results. We sought to remedy that in this paper, using variable-selection methods to help address this issue. Our initial model includes two trauma variables, and three social support variables, as noted in the hypotheses. The first trauma variable refers to childhood. It was constructed as a score by combining the Childhood Trauma Questionnaire (CTQ), indicators of parental drug and alcohol problems, and an indicator of parental death in childhood. Each subscale of the CTQ is assessed to determine whether the score is between 15 and 25, indicating significant trauma. Subscale scores of 15-25 are assigned one point, while subscale scores below 15 are assigned zero points. Each of these five subscale scores of 0 or 1 are added together, and the two indicator variables are added to the scores. In total this variable could range from 0 (none of these experiences) to 7 (all of these experiences). The adulthood trauma variable is the score from the Life Events Checklist. The checklist contains 27 items and includes questions about topics such as

whether the respondent had an immediate family member die, experienced a firing or unemployment, was exposed to combat, was expelled from school, was ever assaulted, and so forth. The three social support variables included in the model are averages across a set of questions asking respondents whether they receive social support from their spouse, their family members (other than a spouse), and their friends. A score of 4 indicates the highest level of support. The initial spectrum of variables from which we applied model selection also included interaction terms of trauma and social support.

The confounders include sociodemographic variables of age (treated continuously), marital status (married/not married), sex (male/female), exercise (present/absent), smoking (never smoker/ever smoker/current smoker) and drinking behaviors (ever drinker in last 30 days/3+ drinks in last 30 days/never drinker in last 30 days), positive affect (scale derived from six items measuring how often in the past 30 days respondents felt cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, and full of life (Mroczek & Kolarz, 1998)), psychological well-being (scale derived from three items), and mastery (scale measuring sense of control from a combination of 12 items measuring perceived constraints and personal mastery). Potential biomarker confounders were also included based on reported associations with the dependent variables in the literature. These included body mass index (BMI), A1c (a measure of diabetes;%), two measures of cholesterol (HDL and LDL; mg/dl), insulin (uIU/ml), and a combined indicator of the number of health conditions/uses of medications

that may affect cortisol levels. A1c, HDL, LDL, and insulin were all measured as part of the biomarker study (Project 4). Health conditions/medications relating to cortisol were reported across Projects 1 and 4, and include thyroid disease, use of hormonal contraceptives, use of prescription hormone therapy, use of stimulant medications, cirrhosis/liver disease, and use of corticosteroids.

Data Analysis

As noted above, cases with missing values on the dependent variables were dropped from the analysis. The few retained cases with missing data in independent variables were processed using dummy variables to indicate missing data. Final models were constructed using significance combined with the change-in-estimate variable selection routine (Maldonado & Greenland, 1993), which are common approaches in public health. First, all predictors were considered and only those that were significantly related to the outcomes ($p<.05$) were retained. Next, each predictor variable was added back into the model to evaluate if it impacted the key coefficient in the model by 10% or more (Maldonado & Greenland, 1993). After variable selection each of the models was relatively parsimonious.

Model 1 (hypothesis 1), estimated using OLS, tested for a linear relationship between the model-selected parameters of female, age, insulin, number of adult traumatic events reported, and family support with logged cortisol as the dependent variable. Model 2a (hypothesis 2) produced odd ratios through use of logistic regression to predict the risk of high CRP. Being

female, BMI, A1c, smoking, and health conditions associated with cortisol levels were the model-selected independent variables for this model. Model 2b (hypothesis 2) used multinomial logistic regression to analyze the relationship between the independent variables of insulin, health conditions that affect cortisol, smoking, age, and BMI with number of chronic health conditions. The dependent variable categories of one health condition and two or more health conditions were compared to the category of no health condition. All data analyses were completed in Stata 13 (StataCorp, 2013).

Results

Key descriptive statistics for the sample are shown in Table 1 and A1. The mean age of respondents was 55.08, and 54% of the sample reported being female. The mean body mass index (BMI) was 28.90, which falls into the overweight category, and the mean A1c was 5.98%, which is indicative of pre-diabetes. Approximately 45% of the sample had ever smoked, and approximately 14% of the sample was smoking at the time of data collection. In terms of health conditions that could affect cortisol levels, the mean number was 0.27, with a range of zero to three. The mean number of adult traumatic events reported by respondents was 1.75 with a range from zero to nine, and mean family support was 3.54 on a scale from one to four, indicating a high level of social support from family and relatively low amounts of traumatic events experienced. In terms of outcomes, the mean logged cortisol level was 2.25, approximately 24% of the sample had high

CRP levels, and for chronic health conditions, 22% reported zero, 30% reported one, and 49% reported two or more.

Table 1 *Descriptive Statistics*

Variables	Mean	*SD*	Range
Female respondent[1]	.54		0-1
Age	55.08	11.76	34-84
Log of cortisol (nmol/L)	2.25	0.59	-1.27-3.66
BMI	28.90	5.77	14.99-60.39
High CRP (>=3&<10) ug/mL)	0.24		0-1
Adult trauma	1.75	1.67	0-9
Family support	3.54	0.60	1-4
A1c (%)	5.98	0.90	3.8-15.2
Smoking			
Ever smoker	.45		0-1
Current smoker	.14		0-1
Insulin	12.65	12.35	1-231
Health conditions assoc. w/cortisol	.27	.52	0-3
Number chronic health conditions			
None	.22		
1	.30		
2 or more	.49		
N	973		

[1]For proportions standard deviations are not reported because the whole distribution is represented.

Model 1 (hypothesis 1) tested for a linear relationship between the model-selected predictors (from variable selection procedures) of female,

age, insulin, adult trauma, family support, and logged cortisol. Table 2 shows the results, with a weak R^2 value of 0.0703 for the model, which indicated that only about 7% of the variation in the dependent variable was explained by the predictors. All of the predictor variables displayed a significant relationship to cortisol, except family support. Each additional year of age was associated with higher logged cortisol (B = 0.01, p<.001), and being female (B = -0.15, p<.001) was associated with lower logged cortisol. Higher insulin (B = -0.004, p<.05) was associated with lower logged cortisol, while greater experience of adult trauma (B = 0.03, p<.01) were associated with higher logged cortisol. This model provides some evidence that trauma is associated with greater inflammation.

Table 2 *Coefficients from Linear Regression Analysis Predicting Log of Cortisol*

Model 1. Logged Saliva Cortisol (nmol/L)	*B*	*SE B*
Female respondent	-0.15	0.04***
Age	0.01	0.002***
Insulin	-0.004	0.001*
Adult trauma	0.03	0.01**
Family support	0.04	0.03
Constant	1.66	0.14***

Note. N = 973. R^2 = 0.0703. Female represents the female slope for the sex variable.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Model 2a (hypothesis 2), shown in Table 3, used logistic regression to determine which model-selected parameters were significantly related to risk of high CRP. Results indicated that women (OR = 1.98, p<.001), those with higher BMI (OR = 1.14, p<.001), those with higher A1c levels (OR = 1.34, p<.01), and current smokers (OR = 1.99, p<.05) all had higher odds of high CRP. The weak pseudo-R^2 (0.1249) suggests a better fit of this model compared to Model 1.

Table 3 Models 2a and 2b.
Odds Ratios and 95% Confidence Intervals from Binary Logistic Regression Analysis Predicting High CRP and Relative Risk Ratios and 95% Confidence Interval from Multinomial Logistic Regression Analysis Predicting Chronic Health Conditions

Model 2a. High CRP	OR	95% CI
Female respondent	1.98***	1.40-2.80
BMI	1.14***	1.10-1.17
A1c	1.34**	1.13-1.59
Smoking		
Ever smoker	1.40	0.92-2.12
Current smoker	1.99*	1.14-3.49
Health conditions associated w/cortisol	1.31	0.97-1.77
Constant	0.001***	0.0001-0.002
Model 2b. Chronic health conditions	**RRR**	**95% CI**
One chronic health condition		
Insulin	1.04**	1.01-1.07
Health conditions associated w/cortisol	1.85**	1.20-2.84
Smoking		
Ever smoker	0.77	0.47-1.24

Current smoker	0.50*	0.26-0.95
Age	1.02	1.00-1.03
BMI	1.03	0.99-1.08
Constant	0.06**	0.01-0.39
Two or more chronic health conditions		
Insulin	1.04**	1.01-1.07
Health conditions associated w/cortisol	2.35***	1.56-3.54
Smoking		
Ever smoker	1.05	0.67-1.66
Current smoker	0.83	0.46-1.49
Age	1.05***	1.04-1.07
BMI	1.07**	1.03-1.11
Constant	0.01***	0.003-0.04

Note. N = 973. Pseudo R^2 = 0.1249 (Model 2a). Pseudo R^2=0.0699 (Model 2b). Female represents the female slope for the sex variable. * p < .05. ** p < .01. *** p < .001.

Model 2b (hypothesis 2; Table 3) used multinomial logistic regression to test for model-selected parameters (identified in variable selection routine) that were statistically significantly associated with number of chronic health conditions. Results indicate that when compared to those with no health conditions, insulin (RRR = 1.04, p<.01) and more health conditions related to cortisol (RRR = 1.85, p<.01) were associated with higher risks of having one chronic health condition, whereas being a current smoker was associated with lower risks (RRR = 0.50, p<.05). Compared to those with no conditions, insulin (RRR=1.04, p<.01), age (RRR=1.05, p<.001), BMI (RRR

= 1.07, p<.01), and health conditions related to cortisol (RRR=2.35, p<.001) were associated with higher relative risk of having two or more chronic health conditions. The weak pseudo-R2 (0.0699) suggests a better fit of Model 2a as compared to Model 2b, albeit both provide a weak explanation of the variation in the outcome variable by the predictors.

Discussion

Many people will experience trauma at least once in their lifetime. This presents a public health problem, as the consequences of traumatic stress influence several aspects of human health. The biological and physiological processes involved are complex, with the social context of individuals potentially influencing reactions to stress. For this study, two hypotheses were tested to examine the role of trauma and social support on the health of individuals. The first hypothesis was weakly supported as the main effect of trauma in adulthood was positively predictive of logged cortisol, but family support was not associated with logged cortisol. Alternatively, our second hypothesis was negated. Overall, trauma and social support do not appear to be related to risk of high CRP or number of chronic health conditions.

In testing hypothesis 1, it was expected there would be a negative association between social support and baseline cortisol levels. In the OLS model we found trauma experienced in adulthood was positively related to logged cortisol (p<0.05). Interestingly, family support was not significant in predicting cortisol levels in the presence of trauma suffered in adulthood and none of the interaction terms tested were significant for trauma and social

support. Instead, it appears social support is a confounder as it impacted the main predictor variable (sex; being female) by 10% or more (Lee, 2014). Further analysis to ascertain if it is a mediator or moderator was inconclusive as model estimates were imprecise in this sample. As no significant predictive relationship was shown between any form of social support and logged levels of baseline cortisol, the first hypothesis was only partially supported (for the trauma-cortisol relationship).

Our findings for hypothesis 1 were consistent with some studies and inconsistent with others. Pico-Alfonso et al. (2004) found baseline waking cortisol unchanged in women victims of intimate partner violence, noting that it was also difficult to determine if cortisol levels were due to another factor. In another study, Johnson et al. (2008) found lower cortisol levels associated with chronic abuse but higher levels associated with PTSD, suggesting further uncertainty as to what was influencing the increased levels. In a similar study, Inslicht et al. (2006) found increased cortisol with a lifetime history, current or remitted, of PTSD. The discrepancies may be explained by certain factors, such as length of time since the traumatic experience, living situations of the participants, and resilience. It may also depend on the severity of the experience and the length of time a person has negative consequences as a result. As this study included individuals affected by several different kinds of trauma, it would help to test the relationship by each type of trauma. Individual perceptions of what trauma and social support are can also influence or inhibit support-seeking behaviors.

Isolation, homelessness, and restricted access to health services are some of the social consequences of experiencing trauma. Social support has been shown to aid individuals in dealing with traumatic experiences, but many of those affected report having a limited social network. For the models of hypothesis 2, we expected to find a relationship between outcomes of CRP and number of health conditions and a higher trauma count and less social support. Findings negated the hypothesis as no trauma (adult- or childhood), social support (friend, family, spouse), nor an interaction of trauma and support were significant or greatly affected the anchoring predictor variable. Again, individual perceptions of what social support is, as well as the ability to identify one's own perceived stress in the self-report measures, may have influenced accuracy of reported information. Another possible explanation may have been that our sample reported a low trauma count, with an average number of events of 1.75 for the adult trauma scale and 0.63 for the childhood trauma scale. Further, 62% of the MIDUS sample reported no childhood trauma and 23% reported only one event. Social support was also high in this sample, as indicated in the results and Table 1. Therefore, the MIDUS sample characteristics may prohibit the extrapolation of broad conclusions to the general American population for trauma and social support.

The current study has several limitations. The complex processes of biological and physiological activity are difficult to measure with constructed measures of social support. Social support can be more efficiently measured

with an improved definition that describes it as a multi-dimensional construct. The support variables were constructed by measures of self-reported family, friend, and spousal support. They do not include other forms of support like church or support groups, which may have exerted a greater or different impact. Further, the measure for trauma count was constructed by combining several types of traumatic experiences, but it does not assess whether or not the individuals sustained post-traumatic stress from the experiences. Relatedly, the social support variables do not have a time element, and so timing of the support in relation to the trauma was not modeled. Therefore, effects from early or late support could not be teased out and would presumably have an impact. Studies would benefit from exploring these health problems in chronic trauma survivors in comparison to this study population that features a lesser history of trauma. In addition, the variable selection approach where multiple domains (e.g., social, socioeconomic, etc.) were analyzed simultaneously may have led to the discovery that previously significant predictor variables in the literature were more likely confounders of other predictors than predictors of health-associated outcomes. Finally, the data were collected from 2004-2006, and more than a decade has since passed. Although this is the most relevant source of data for this study, the experiences of older adults today may be different. For example, since the collection of the data, older adults have lived through the Great Recession, which was accompanied by a loss of savings and a foreclosure crisis for many Americans (Hurd & Rohwedder, 2010), both of

which may have been significant traumatic experiences (Cagney et al., 2014; Yilmazer, Babiarz, & Liu, 2015) and may have also affected social support relationships. Additionally, the older adult population is becoming more diverse, and there is reason to believe that new models of diversity and health disparities are necessary (Hsu et al., 2018). Thus, the generalizability of the findings to the contemporary older adult population may be limited.

Conclusion

The effects of trauma on human health are complex. Testing for relationships between biomarker data and constructed measures of social support, among known and possible confounders, can improve estimates to provide better clarification of relationships and interpretation of results. Narrowing down the actual predictors and teasing them out from the confounders is important for developing public health policy and plans, as well as providing effective clinical treatments. Using data from MIDUS 2 and variable selection methods to improve on past research, this study found trauma experienced in adulthood was positively related to logged cortisol ($p<0.05$), in support of hypothesis 1, but family support was not a significant predictor of cortisol levels in the presence of trauma suffered in adulthood. There was no support for hypothesis 2; trauma (adult or childhood) and social support were not associated with high CRP or number of health conditions, in contrast to the hypothesis. As studies continue to explore this topic and provide suggestions for further research, continued interdisciplinary approaches, such as those in this study, would greatly add

to the available information and understanding of the problem. Collaboration between health and social service providers can be improved by the addition of constructs and findings from both areas, making treatment and outcomes better for affected individuals.

Conflict of Interest to Declare

The authors have no conflicts of interest to disclose.

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Appendix Table 1. Descriptive Statistics—Sample Means and Percentages for All Examined Predictors, Outcomes, and Confounders, MIDUS

	Analytic Sample		Biomarker Sample	
	Mean (SE)	**Range**	**Mean (SE)**	**Range**
Female	53.5%		54.7%	
Married	72.7%		72.2%	
Body mass index at Wave 2	28.90 (5.77)	14.99-60.39	29.18 (6.01)	14.99-60.39
Adult trauma	1.75 (1.67)	0-9	1.80 (1.73)	0-9
A1C (%)	5.98 (0.90)	3.8-15.2	5.99 (0.92)	3.8-15.2
HDL Cholesterol (mg/dl)	54.88 (17.74)	19-121	54.63 (17.60)	19-121
LDL Cholesterol (mg/dl)	106.06 (34.74)	16-283	106.31 (35.15)	16-283
Insulin	12.65 (12.35)	1-231	12.78 (12.32)	1-231
High CRP	24.3%		24.8%	
Cortisol	11.14 (6.64)	0.28-38.93	13.11 (59.52)	0.34-1889.31
Health conditions associated w/cortisol	0.27 (0.52)	0-3	0.28 (0.52)	0-3
Age at time 2 (years)	55.08 (11.76)	34-84	55.26 (11.78)	34-84
Smoking				
Never smoker	40.9%		40.8%	
Ever smoker	44.8%		44.6%	
Current smoker	14.3%		14.6%	
Alcohol consumption at time 2				
Ever drinker, last 30 days	44.6%		44.4%	
3+ drinks/wk, last 30 days	23.8%		22.9%	
Never drinker, last 30 days	31.6%		32.7%	
Exercise at time 2	79.9%		78.8%	
Number of health conditions				

0	21.7%		21.2%	
1	29.8%		29.1%	
2+	48.5%		49.7%	
Family support	3.54 (0.60)	1-4	3.54 (0.60)	1-4
Friend support	3.30 (0.68)	0-4	3.33 (0.64)	1-4
Spouse support	2.82 (1.58)	0-4	3.62 (0.53)	1-4
Total child adverse events	0.63 (1.03)	0-6	0.63 (1.03)	0-6
Positive affect	3.45 (0.69)	1-5	3.44 (0.70)	1-5
Psychological well-being	9.11 (1.75)	3-12	9.12 (1.74)	3-12
Mastery	5.65 (0.96)	1.08-7	5.64 (0.96)	1.08-7
N	973		1054	